

CLINICAL AND MORPHOLOGICAL CORRELATIONS OF LIVER CIRRHOSIS

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Summary: Liver cirrhosis is a chronic, polyetiological, progressive liver disease that is characterised by a significant decrease in the number of functioning hepatocytes, increasing fibrosis, rearrangement of the normal structure of the parenchyma and subsequent development of liver failure and portal hypertension..

As part of additional diseases, cardiovascular pathologies and DM 2 -34.3% take the leading place.

Keywords: Liver cirrhosis (LC), liver diseases, Pathogenesis, clinical presentation, immune mechanisms, Splenomegaly, cytolysis, hepatocytes, blood circulation, liver transplantation, coma, alcoholic cirrhosis. Parenchyma.

Liver cirrhosis (LC) represents the final common pathway of all chronic liver diseases. Despite the availability of efficacious etiotropic therapeutic modalities in contemporary medicine, the prevalence and mortality rates associated with CKD and its complications remain high and continue to increase. In economically developed countries, CKD represents one of the six leading causes of mortality among individuals aged 35-60 years, with a prevalence ranging from 14 to 30 cases per 100,000 population.

PATHOGENESIS

1. Action of etiological factors: cytopathogenic action of viruses, immune mechanisms, influence of hepatotoxic cytokines, chemokines, prooxidants, eicozonoids, acetaldehyde, iron, lipid peroxidation products.
2. Activation of Ito cell function, which leads to overgrowth of connective tissue in Dysse spaces and pericellular fibrosis of the liver.
3. Disturbance of blood supply of liver parenchyma due to capillarisation of sinusoids and reduction of vascular channel with development of ischemic necrosis of hepatocytes.
4. Activation of immune mechanisms of cytolysis of hepatocytes.

At bridge necrosis of hepatocytes, T-lymphocytes converge to the lesion zone, which activate Ito cells synthesising type I collagen, which leads to fibrosis. In addition, microscopically, false lobules without a central vein are formed in the hepatic parenchyma.

CLINICAL PRESENTATION

The clinical presentation of cirrhosis is characterised by a constellation of general symptoms, including weakness, reduced ability to work, abdominal discomfort, dyspeptic disorders, increased body temperature, joint pain, flatulence, pain and heaviness in the upper half of the abdomen, weight loss, asthenia.

An examination of the liver reveals its enlargement, accompanied by thickening and deformation of the surface, and sharpening of the edge. Initially, there is a uniform moderate enlargement of both lobes of the liver. Subsequently, in the majority of cases, the left lobe is the predominant enlargement. Portal hypertension is evidenced by a moderate enlargement of the spleen.

CLINICAL AND MORPHOLOGICAL CORRELATIONS (1)

Ocular symptoms. Eyelid retraction and lagging of the upper eyelid from the eyeball are more frequently observed in cirrhotic patients compared to the general population. There are no signs of thyroid disease. Serum free thyroxine levels are normal.

Enlarged parotid salivary glands and Dupuytren's contracture **may also occur in alcoholic cirrhosis.**

CLINICAL AND MORPHOLOGICAL CORRELATIONS (2)

Steatorrhea is common even in the absence of pancreatitis or alcoholism. It may be due to decreased secretion of bile acids by the liver.

Splenomegaly and dilated venous collaterals on the anterior abdominal wall usually indicate portal hypertension.

Abdominal wall hernias develop frequently in ascites. They should not be radically treated unless they are life-threatening or the ascites is not adequately compensated.

CLINICAL AND MORPHOLOGICAL CORRELATIONS (3)

Primary liver cancer is a frequent complication of all forms of cirrhosis except biliary and cardiogenic cirrhosis.

Biliary stones on ultrasound (usually pigmented) have been found in 18% of men and 31% of women, which is 4-5 times more common than in the population. The presence of stones does not affect survival.

A low ratio of bile acids to unconjugated bilirubin and a very high level of monoconjugated bilirubin in bile predispose to the development of pigmented stones.

CLINICAL AND MORPHOLOGICAL CORRELATIONS (4)

Cardiovascular lesions. Patients with cirrhosis develop atherosclerosis less frequently than in the general population, myocardial infarction occurs almost 4 times less frequently than in persons without cirrhosis. In patients with liver cirrhosis, belonging to group C according to Child, the content of nitric oxide in exhaled air is 2 times higher than in healthy people.

Kidney damage. In all forms of liver cirrhosis, blood circulation in the kidneys is impaired. In particular, the blood supply of the cortical layer deteriorates, which contributes to the development of hepatorenal syndrome. On the background of chronic hepatitis C, cryoglobulinaemia and membranoproliferative glomerulonephritis develop.

CLINICAL AND MORPHOLOGICAL CORRELATIONS (5)

Infectious complications. In liver cirrhosis, phagocytic activity of reticuloendothelial cells is reduced, which is partly due to portosystemic blood shunting.

As a consequence, bacterial infections (usually caused by intestinal microflora) often develop. These complications occur in 4.5% of cirrhotic patients annually.

The clinical triad of liver cirrhosis comprises three distinct signs: the presence of portal hypertension, a palpable liver with a dense and stony consistency, and the presence of hepatic stigmata.

The complications of liver cirrhosis can be classified into the following categories:

- 1) Bleeding from varicose veins of the oesophagus and stomach
- 2) Hepatic encephalopathy and coma
- 3) Erosions and ulcers of the stomach and duodenum
- 4) Portal vein thrombosis
- 5) Cirrhosis-cancer
- 6) Ascites-peritonitis
- 7) Hepatic nephropathy (hepatorenal syndrome)

CONCLUSION: The prognosis of the disease largely depends on the causes of cirrhosis development, the speed of final diagnosis, the severity of the disease, available vital reserves (presence of chronic comorbidities) and timely medical care.

Statistically, the ten-year survival rate ranges from 35% to 67%. It is predicted that with 'C' class of severity (more than 10 points on the Child-Pugh scale), the probability of death within one year is 50%.

The model of end-stage liver disease (MELD) is used to estimate the expected life expectancy for liver transplant planning.

Three-month MELD statistical prognosis:

- Less than 9 points - 2.9% mortality;
- 10-19 points - 7.7%;
- 20-29 points - 23.5%;
- 30-39 points - 60%;
- over 40 points - 81%.

After liver transplantation, the five-year survival rate is 80%.

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